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MYCOLOGIA

VOL. VII JANUARY, 1915 NO. 1

A TIMBER ROT ACCOMPANYING HYMENOCHAETE RUBIGINOSA (SCHRAD.) LEV.

(WITH PLATES 149–151, CONTAINING 30 FIGURES)

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The Hymenomycetes among the fungi play the leading rôle in the disintegration and destruction of wood. Since Theo. Hartig's first attempt at the scientific investigation of the decay of wood in 1833,[8] other workers have contributed from time to time and as a result an extensive literature on this phase of mycology has been gradually collected.

Of the investigated forms, the Polyporaceae have been most extensively studied. This is due in part at least to their large, conspicuous fruit bodies and the obvious relation between them and the decay resulting in the wood. Many other Basidiomycetes as well as Ascomycetes are wood-inhabiting, and interesting results have already been obtained from studies of isolated forms in various families. The field of research may be divided into two lines of effort; to secure definite data on the chemical composition of wood in general and of changes resulting from decay, and to augment our present field of knowledge of the decay of wood by a study of uninvestigated wood-inhabiting forms. The present study of *Hymenochaete rubiginosa* (Schrad.) Lév. was undertaken with the latter point in mind.[1]

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[1] This work was carried on in the Botanical Laboratory of Cornell University under the direction of Prof. Geo. F. Atkinson, to whom the writer is indebted for the suggestion of the problem and for advice.

[MYCOLOGIA for November, 1914 (6: 273–323), was issued December 10, 1914.]

Hymenochaete rubiginosa is a common saprophyte on decorticated chestnut in the vicinity of Ithaca, N. Y., and is also found more rarely on decorticated oak. The brown, resupinate or dimidiate fruit bodies (Figs. 1, 3), rarely over four or five centimeters in diameter, are usually to be found where oak and chestnut timber lies on the ground in the forest. Chestnut-rail fences which extend into the deep shade of the woods present an ideal place for its collection and almost invariably the lowest rail, partially immersed in leaf mold, is infested with the fungus. Where the rail is oblique to the surface of the ground and one end is imbedded in the humus, thereby insuring a sufficient supply of moisture, the fruit bodies may be entirely resupinate and reflexed. On fallen trunks they usually become dimidiate and often hollow-ungulate, the pilei imbricated, irregularly united and sharply oblique to the substratum. When mature, the shelving pilei are smooth above, except for the pubescent margin, and exhibit fine concentric lines on their upper surface. These are not the result of perennial growth, since the fruit bodies are annual, but rather of fluctuations in seasonal growth brought about by varying amounts of moisture. The fruiting surface, or hymenium, of the pilei is glabrous at maturity except at the margin. When viewed with a pocket lens of ten diameters, it is seen to be covered with many brown setae which project upward from the grayish hymenium. It is due to this character that Léveillé[10] first separated such forms from the genus *Stereum*.

Development of the Fruit Bodies.—As already noted, the fruit bodies make their appearance on decorticated wood. Even in collecting several hundred specimens at periods extending over two years, no exceptions were found to the above rule. The fruit bodies rarely appear on wood in an advanced stage of disintegration. A few cases were observed where the wood was badly decayed to a depth of a centimeter under the fruit bodies due to attacks of this or other fungi. In such cases, however, the fruit bodies were always connected with the firm, hard wood in the core of the log. They are never formed from mycelium in wood which has lost its firmness or exhibits advanced stages of decay.

The first evidence of the formation of fruit bodies is the appearance of small, brown, mycelial wefts on the surface of the infected wood. These are usually discoid in shape and about the size of the head of a pin. More rarely, narrower brown areas occur which are elongated lengthwise of the log. In such cases the hyphal wefts arise from seasoning checks in the wood; the first case is by far the most frequent. Usually several, or as many as a dozen, of these small brown disks arise in the area of a square inch.

The manner of growth of the brown disks, once formed, depends on their position with reference to the substratum. If they occur laterally on a log, dimidiate fruit bodies are formed; if on the under side, wholly resupinate forms arise. In either case, growth goes on all along the margin of the disk for a time. Subsequently, in the dimidiate forms, the upper margin continues its growth, arches out away from the substratum, and forms the pileus. The lower margin continues to expand for a short time downward along the substratum and may unite with other fruit bodies, but eventually, and much sooner than the arching upper margin, ceases to grow. The final result is a cluster of shelving fruit bodies, partially coalesced behind next to the substratum, but with separate free outer margins.

The development of the wholly resupinate forms is easier to follow. Growth is much more regular along the margin and may continue so evenly that circular fruit bodies are formed. Usually, however, owing to the irregularities of the substratum, growth is restricted here and there so that the contour of the fruit bodies becomes irregular and sinuous. This appearance is still further enhanced because the fruit bodies often coalesce (Fig. 21). Two advancing margins meet and the hyphae intermingle. The line of union remains distinct for a time as a pubescent ridge through the hymenium, but eventually the latter covers this and the line of delimitation between the two fruit bodies is obliterated. In this way, extended hymenial layers are formed in the resupinate type. Evidence of their synthetic origin may be observed, however, by carefully removing them from the substratum and examining the surface next to the wood. In such a case, one finds as many points of attachment as there are units entering into the structure of the compound sporophore.

Age of Fruit Bodies.—The fruit bodies of *Hymenochaete rub-*

iginosa are annual. Growth may go on at the margin until well into the autumn and as a result an increase in surface area occurs. but in no case was a new hymenial formation found the following spring. After the first season, the hymenial surface loses its rich rubiginous color and becomes dull and grayish-brown. As the fungus dries out, as happens in many cases, extensive checking occurs. The fruit bodies of the season are readily discernable from those of former years because of the differences enumerated above.

The fungus is essentially a xerophyte. It withstands to a remarkable degree the many vicissitudes of climate and recovers well after a prolonged drought. Within a few hours after precipitation, spore formation begins again and continues as long as favorable conditions prevail. Interrupted spore formation may go on in this way during the summer and autumn.

The time at which fruit bodies first begin to shed spores varies with conditions. Fruit bodies with a surface area of a square centimeter were found to be producing viable spores, and it is possible that spore formation may have begun when they were much smaller. Spore formation begins before the fruit bodies have attained their ultimate size and continues with many interruptions until late autumn.

Structure of the Fruit Bodies.—There are three distinct layers discernable (Fig. 18), which give to the fruit body a stratose structure. The lower exposed layer bears the hymenium on its outer surface and makes up the greater part of the thickness of the fruit body (Fig. 4) and its limits of variation are such that a fruit body may be fully twice as thick in some places as in others. The second layer consists of a narrow stratum of hyphae near the upper surface, closely entwined and fastened together. The third and last is a loose, floccose stratum of hyphae, in which the course of the filaments as individuals may be readily followed. The last two layers exhibit little variation in thickness.

The hymenium of the species under consideration (Fig. 19) consists of three distinct structures, viz.: large and prominent brown cystidia (*a*), colorless basidia (*b*), and a third element similar to the basidia, but shorter and only slightly enlarged at the tip (*c*). These may be immature basidia which subsequently elongate and produce spores. This conclusion is plausible because spore formation continues for a long period and careful examination of fruit bodies during the summer and autumn has shown that the spores produced by a basidium mature at approximately the same time.[2] It follows that the basidia probably develop successively during moist periods, a condition which accounts for the continued spore formation.

The large, brown setae vary in shape from conical-acute to bluntly cylindrical and measure 70–100 μ by 4–5 μ. Below, they taper gradually into thin-walled hyphae which extend horizontally. Massee[11] makes note that in addition to the normal setae, stout cylindrical, obtuse, thin-walled, pale-brown bodies, intermediate between setae and cystidia, are sparingly met with in the hymenium. A careful microscopical examination of a number of specimens failed to reveal such structures in the form common about Ithaca, N. Y.

The basidia are approximately one third the length of the cystidia and are quite colorless. They are attached below to brown hyphae.

The spores are hyaline, ellipsoidal, and measure 5.5–6.4 μ long by 2.8–3 μ broad (Figs. 2, 17^a). These dimensions conform closely to the spore measurements by Massee, who gives them as 5 × 3 μ for this species. Saccardo[14] in his description says "sporis cylindraceis, curvatis, 5–6 μ long." The spores of specimens found about Ithaca correspond in length, but are neither cylindrical nor curved. Spores taken from a number of fruit bodies all proved to be ellipsoidal.

Spore Germination.—Spores were obtained from many different specimens through a period of two years. The fruit bodies as brought in from the field were placed in moist chambers over sterilized petrie dishes, and the spores obtained in this way were preserved for further study.

Spore germination was attempted in Van Tieghem cells which had been carefully sterilized previously with 5 per cent. corrosive sublimate solution and rinsed in distilled water. Mounts were then made, using tap water, distilled water, filtered chestnut wood extract, prune decoction and a solution of beef extract and malt.[3] The best germination was obtained with chestnut wood extract. Slight germination occurred in tap water and in the malt beef extract; the others gave negative results.

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[2] H. M. Ward[18] has described and figured similar structures in the hymenium of *Stereum hirsutum* Fr. and has suggested further that these intermediate hyphae may grow forward to develop a new hymenial layer.

Before germination, or with its inception, the spore becomes vacuolate (Figs *b* and *e*). Then germination occurs at one or both ends. Lateral germ tubes were not observed. A small papilla, always of smaller diameter than the spore, is formed, which gradually elongates to form the germ tube. At the end of forty-eight hours, some of the germ tubes attained a length of from four to six times the length of the spore (Fig. 8). Thereafter growth was very slight. In preparations a week old, here and there a germ tube had branched, but in general only slight elongation occurred after the second or third day. Mycelial wefts were not obtained in hanging drop cultures. Rarely one or two cross walls were formed in the germ tube, but these were never accompanied by clamp connections.

An earnest effort to secure pure cultures of the fungus led to no tangible results. Various media were tried, among which may be enumerated prune agar, bean agar, chestnut wood decoction agar, chestnut wood decoction gelatin, corn meal moistened with wood decoction, corn meal moistened with tap water, sterilized chestnut wood, sterilized oak wood, bean tubes, potato tubes, carrot tubes, and a medium made after Marpmann's formula.[4] Inoculations were made with newly fallen spores and with germinating spores from hanging drop cultures. A sparse, floccose mycelium from germinating spore infection was formed in the bean tubes, but the hyphae were always restricted in their growth. Attempts to transfer these colonies to more favorable media resulted in failure. New experiments are now under way with this object in view, and it is to be hoped that they may be more fruitful of results. The timber rot accompanying the fruit bodies of this fungus on chestnut and oak, compared with the condition of the normal wood, may now be described.[5]

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[3] 2½ gm. Liebig's beef extract[13], 2½ gm. Loffund's malt extract, 100 c.c. water.

[4] Dissolve by cooking 10 gm. of gelatin and 10 gm. of agar in 500 gm. of beef extract; then add to the solution 10 gm. glycerine, 10 gm. salt, 5 gm. ammonium phosphate, and 4 gm. potassium nitrate; filter.

Character of Normal Wood.—The wood of chestnut is of the ring-porous type.[12] There are several rows of large vessels in the spring wood out of which branching rows of smaller vessels extend radially into the summer wood. The transition from large to small vessels is usually very abrupt (Fig. 6) and the radial arrangement of the small vessels is often somewhat obscure. Annual ring formation is pronounced, each ring being sharply delimited from the others. The width of the ring varies within wide limits; sometimes in coppice growth it is over half an inch in thickness. The pith rays are minute and scarcely distinguishable with the naked eye.

Miscroscopically the wood of chestnut is seen to consist of (*a*) uniseriate pith rays, (*b*) pitted vessels, (*c*) metatracheal parenchyma with simple or semi-bordered pits, (*d*) tracheids and (*e*), wood fibers. The last are not typical fibers, but of the nature of fibrous tracheids.[6] The vessels are discernible in cross section by their size. Fibrous tracheids, tracheids, and wood parenchyma look much alike; the last may be distinguished, however, through its protoplasmic contents. The vessels exhibit the greatest variation in size in the annual ring. The cell lumina of the parenchyma and prosenchyma are somewhat wider in the spring wood than in the summer wood, but show no great range of variability in actual size.

Description of Decay in Chestnut.—The first evidence of incipient decay in chestnut wood is the appearance in the wood of irregular areas in which the tissues have lost their natural brown coloration and become grayish-white (Fig. 5, 10). These areas are 1 mm. or less in cross diameter by 5–25 mm. in longitudinal direction. The wood between the lighter areas remains as sound apparently as ever.[7] Logs, limbs an inch or more in diameter, rail fences, chestnut posts and structural chestnut timber may be attacked. The infection at the start is a local one, and the diseased areas first occur in the last few rings. In such cases they are next to the ground, or with large logs on the lateral side most protected from drought. Only in an advanced stage is the whole cross section attacked, and before this condition is reached the inroads of the fungus are usually arrested through lack of moisture. Chestnut is very durable in contact with the soil, and it is not uncommon to find fallen decorticated limbs and branches showing the decay described above on the lower side, while they are as sound at the core and on the upper side as normally. Only where sufficient moisture is available is the center of the log attacked. Cracks due to checking and frost action greatly facilitate the action of the fungus, since they permit the ready ingress of water into the deeper-lying tissues (Fig. 28).

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[5] Direct evidence of the causal connection of *Hymenochaete rubiginosa* with the peculiar rot which accompanies it, has not been obtained in the present study. It is reasonable to assume that the rot in question is caused by this species since it always accompanies this form on chestnut. Further, the same type of rot is associated with this fungus on oak. This evidence, though not conclusive, leads to the inference that this peculiar decay of oak and chestnut is caused by *H. rubiginosa*.

[6] In length and taper, fibrous tracheids resemble fibers; in width and bordered pits, tracheids.

All parts of the annual ring are susceptible to the attacks of the fungus (Fig. 14). The white areas of varying extent and irregular outline may include within their boundaries one or more vessels in the spring wood, or be entirely confined to the outer portion of the ring. The last is usually most severely attacked, however. Occasionally infected spots coalesce and form large areas, although they usually remain free from the start.

At first there is no disintegration in the wood other than the formation of the irregular white areas. The elements retain their original size, thickness, and continuity with one another. Subsequently near the center of the white areas a small cavity appears (Fig. 6). This is bounded at first by white tissue on all sides and includes but a small portion of the diseased area. The white tissue at the margin remains undisturbed.

Within the cavity itself, disintegration is not complete. It is filled with long, white, fibrous elements which remain loosely attached or entirely free from one another. These are thick-walled and offer great resistance to the dissolving action of the fungus. The cavities gradually enlarge as the disintegration goes on until finally all that remains of the original white area is a narrow boundary line about an enlarged cavity filled with isolated or loosely bound white fibers (Fig. 12). The wood between the cavities retains its original color and is, to all appearances, as sound as ever. In age the white contents and lining of the pockets may disappear and the wood presents the appearance of Figure 9.[8]

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[7] The whole mass of tissue is never affected as in the case of some forms 12, 16, and the wood never loses completely its firmness and elasticity.

In order to trace the progress of the decay in the wood, the following methods were employed. Diseased wood was cut into blocks of convenient size, which were then boiled to fix the hyphae. The air remaining in the tissues was exhausted with an air pump and the blocks were finally imbedded in celloidin in the usual manner. Sections 10 microns thin were used.[9]

A microscopical examination of the wood reveals the action of the fungus to better advantage. In the infected areas the hyphae run vertically in the cell lumina and are closely applied to the tertiary wall. In cross section (Figs. 22 and 26) they appear as small black dots within the cell cavity. Viewed in longitudinal section each element is seen to contain from several to many hyphae which extend vertically from cell to cell and penetrate the walls at will. Preference is shown for the pits on the walls as in the case of some other forms (Figs. 27 and 29), but the avenues of penetration are by no means restricted to the pits. The hyphae pass directly through the wall with little or no constriction (Fig. 23). Subsequently the perforations thus arising are further enlarged through enzyme action so that the hyphae appear to pass through openings much too large for them. In an advanced stage of decay only the openings are left as the hyphae disappear (Figs. 25 and 30). Nests of hyphae also accumulate in vessel cavities, but the centers of infection are usually in areas where only parenchyma and fibrous tracheids occur. Where pith rays cross the diseased areas, they are attacked and eventually destroyed.

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[8] Another type of decay was observed in the wood near the surface of the logs which exhibited in the deeper-lying tissues the characteristic decay already described. For a distance of several millimeters inward from the surface, the tissue had turned dark-brownish-black. Whether this decay was caused by the same fungus as that in the deeper-lying tissues was not determined. A species of *Dasycypha* was frequently found accompanying the Hymenochaete and this may have been responsible for the second type of decay.

[9] In preparing sections for microscopic study both temporary and permanent mounts were made. The first were employed to observe through michrochemical reactions the chemical changes in the wood brought about by the fungus. Among the lignin tests which were used may be enumerated the HCl-phloroglucin reaction, the $KClO_3$-HCl-phenol reaction, aniline sulphate and H_2SO_4, and thallium sulphate in equal mixtures of water and alcohol.[19] The cellulose tests included chlor-zinc-iodide, sulphuric acid, I-KI and iodine followed by sulphuric acid.

The sections used for permanent mounts were stained in several different ways, viz.: Delafield's haematoxylin and aniline safranin, Haidenhain's haematoxylin and safranin, Haidenhain's haematoxylin and methyl green, 20 per cent. aq. tannic acid and methyl violet, ruthenium red and methyl green. The last two stains enumerated gave the best results with the preference in favor of the methyl violet.

In staining with methyl violet, the material was first treated with a twenty per cent. aqueous solution of tannic acid for twelve hours. It was then quickly rinsed with water and transferred directly to a one per cent. solution of methyl violet for three minutes. The excess stain was removed with 95 per cent. alcohol and the material was finally cleared in clove oil. Only the hyphae in the tissues retained the stain.

The method pursued with ruthenium red and methyl violet was that recommended by Eisen[4]. It has also been described in a recent paper by Learn[9] and does not require further explanation here because no deviations were made from the prescribed formula.

Chemical Changes in Chestnut Wood.—The first chemical change which is brought about by the fungus is that of delignification. It is due to this action that the elements lose their normal brown color and become white. The fungus probably secretes dissolving enzymes which attack the tertiary layers first and work outward. The compounds which are known collectively as lignin are entirely dissolved and walls of pure cellulose left behind. Near the center of infected areas all stages in the process of delignification are to be found (Fig. 26). In the sound cells just without the diseased tissue, all three cell layers respond to tests for lignin (Fig. 22 *c*), although the tertiary layer appears to be less strongly lignified than the others. The elements nearer the center of infection are already partially delignified (*b*). In some the tertiary layer no longer gives the lignin reaction, or but feebly, while in others the secondary and primary layers have become involved and delignified. The lignin reaction persists longest at the cell corners where the wall is thickest, or about the small intercellular spaces which often occur there, but even here it disappears eventually, and the tissue that remains consists of almost pure cellulose.

As soon as the middle lamella is delignified, the cells separate from one another (Fig. 26 *c*). Whether the middle lamella dissolves or splits is difficult to decide, but it would appear that the former is the case, in that ruthenium red failed to reveal any trace of the middle lamella where the elements had separated. No eroding action of the fungus is to be noted at this stage. The infected area is filled with a mass of white cells, in part free from one another, or loosely joined at the corners where the middle lamella has persisted.

Before the changes above enumerated have taken place in all the outer cells of the infected areas, further alterations usually occur in the elements first infected. The cellulose walls now undergo digestion (Fig. 11), probably in the usual way through the secretion of cytolytic enzymes.[10] The vessels, parenchyma cells and pith ray cells together with the thin-walled prosenchyma are almost entirely dissolved (Fig. 20). The thicker-walled prosenchyma persists the longest, and after the other elements have disappeared remains as a white fibrous structure partially filling and lining the cavities that have arisen. The abundance of these fibers depends in a large part upon the cytolytic activities of the fungus. The dissolving action may go on in extreme cases until the pockets are quite empty of contents, but usually, for reasons which cannot be satisfactorily explained, the activities of the fungus are inhibited before this condition is reached.

The conditions met with in the dark-brown peripheral tissue, previously described, are quite different from those enumerated above. All the cells here have been attacked and partly digested (Fig. 7). Many hyphae are to be seen in vertical view closely applied to the cell walls and the latter fail to respond to either the phloroglucin-HCl or the chlor-zinc-iodide reaction. R. Hartig[7] noted the same condition in his study of *Merulius lacrymans*. It is possibly explained on the supposition that the fungus has dissolved out but a part of the lignin, and that the portion remaining conceals the chlor-zinc-iodide reaction for cellulose. The periph-

[10] Czapek[3] considers lignified walls as made up of a hadromal-cellulose ester. The attacking fungus may secrete two or more enzymes, among which may be included hadromase and cytase. The first splits off the compound ester and removes the hadromal. The cellulose remaining behind is subsequently dissolved by the cytase.

eral decay is always restricted, however, and does not seem to be important.

Condition of the Wood Between the Pockets.—As previously noted, the wood between the infected areas remains unaltered chemically and still responds to the various lignin tests. Here and there in a cross section a fungal filament may be seen extending from cell to cell, but the tissue is usually quite free of ramifying hyphae. In general, delignification occurs only in those tissues where the hyphae extend vertically.

It is obvious that mycelial connection exists between the pockets in the wood. This is brought about in some cases by isolated hyphae which extend horizontally through the tissues. More commonly, however, connecting strands of mycelium (Fig. 24) are formed for this purpose. These, as noted in the figure, consist of a number of filaments closely intertwined and forming a horizontal bridge between the pockets. The strands thus arising extend both radially and tangentially. They cause no chemical alteration in the wood other than that correlated with their penetration, but appear to be a means of spreading the areas of infection horizontally.

Character of Normal Oak Wood.—While closely related botanically to chestnut, oak departs decidedly from the former in the anatomy of its wood. Both possess ring-porous wood with rows of smaller vessels which branch in the outer portion of the ring. The transition from large to small vessels may or may not be decidedly abrupt.[11] Annual ring formation is as pronounced as in chestnut. The chief difference between the two woods consists in the presence of large multiseriate pith rays (Fig. 16) in oak wood accompanying the smaller uniseriate rays. Contrasting the two genera, oak has two kinds of pith rays, chestnut one.

When examined microscopically, the wood of oak is found to consist of the same elements as those of chestnut. There are several anatomical differences worth noting, however. The vessels have thicker walls. The parenchyma in oak is more abundant and makes up a greater proportion of the wood. There is an abrupt transition from tracheids to wood fibers, and fibrous tracheids do not occur. The tracheids possess bordered pits; the fibers narrow, oblique, simple pits.[12] This is in strong contrast to the chestnut where all gradations between tracheids and fibers occur, and the latter are characterized by bordered pits of the usual type. Further, the fibers in oak have thicker walls and are more strongly lignified.

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[11] Abrupt in white oak, gradual in live oaks and red oaks.

In tangential section (Fig. 13) the two types of pith rays are easily discernible. The multiseriate rays appear as broad fusiform structures extending for several millimeters in a vertical direction, while interspersed between them and far surpassing them in number, are the small uniseriate rays. The latter are shorter than those in chestnut.

Description of the Decay in Oak.—The decay of oak associated with *Hymenochaete rubiginosa* is similar to that of chestnut. White areas of varying extent and irregular outline are formed (Figs. 13, 16 *a*), which extend a millimeter or less across the grain, but often a centimeter or more longitudinally. These subsequently give rise to cavities or pockets lined and partially occluded with white fibers. After this stage further disintegration ceases and the wood appears sound except for the presence of many pockets scattered quite regularly through it. The wood of chestnut and oak never becomes soft and badly disintegrated in this type of decay, but the fungus exhibits a remarkable similarity of action on these two hosts.

The effect of the mycelium of *Hymenochaete rubiginosa* on the wood of chestnut and oak is comparable in its grosser aspects to that of *Trametes abietis* Karst. on the red spruce,[1] and the "partridge" wood of oak caused by *Stereum frustulosum* (Pers.) Fr.[13] In each case the areas of disintegration are at first localized. The elements of the wood within the infected areas are wholly or in part dissolved and cavities arise which are lined with a layer of almost pure cellulose, and remains of delignified elements.[14] In the case of *Trametes abietis* the wood between the original areas of infection is finally attacked and broken down so that the whole mass of wood tissue eventually loses its firmness and is in large part destroyed. The same does not apply to the other fungi mentioned since the cells remain intact and sound between the diseased areas so far as can be detected by microchemical reactions.

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[12] It is held by some that all prosenchyma is equipped with bordered pits. The "so called" oblique simple pits in the fibers are interpreted as flattened bordered pits which have been spirally stretched.

[13] Described by R. Hartig[6] as *Telephora perdix*.

[14] Weir[18] has recently published a description of a new fungus, *Fomes putearius* Weir, in which the decay is similar to that of *T. Pini* Fr. The lignin reduction, however, is on a much larger scale and the cellulose pockets are frequently two inches in length and vary in breadth according to the structure of the host.

The similarity between *Stereum frustulosum* and *Hymenochaete rubiginosa* in their manner of attack and effect on the wood is striking. Both are xerophytic fungi and attack decorticated wood which is sound or little decayed through the attacks of other fungi. The first is a perennial form reported on oak alone, so far as I have observed, the second an annual form found on oak, chestnut, and several other hosts. The first evidence of attack in both is in the formation of white areas which respond to the tests for cellulose. Subsequently the elements in these are in part digested and cavities are formed which in one stage of the disease are lined with a white layer of cellulose. The wood remains sound between the diseased areas and in the final stage the condition resulting is comparable to a honeycomb in which the cavities of the wood represent the chambers in the comb, and the tissue lying between, the walls of the chambers. The white lining has entirely disappeared at this stage.

From the preceding paragraph it follows that the decay in oak associated with *H. rubiginosa* is very similar, superficially, to that caused by *Stereum frustulosum.* Closer examination of the specimens at hand, however, has revealed a difference which may be of value in separating these two types of rot. The flecks caused by *Stereum frustulosum* are shorter and wider than those associated with *H. rubiginosa.* In radial view they appear as a rule from oval to elliptical in shape, while those of *H. rubiginosa* are narrow elliptical to long cylindrical. In the final stage of *Stereum frustulosum* the wood is much more porous, due to the large size of the cavities and the small spaces intervening.[15]

Chemical Changes in Oak Wood.—What has been said regarding the method of attack and chemical changes in the wood for chestnut applies equally well for oak. The first visible evidence of attack is the formation of white areas in the tissue due to the delignification of the elements. The centers of infection lie between the broad pith rays. In the white spots the hyphae run vertically and become closely applied to the walls of the cells. The fungus works from the lumen outwards, and first removes the lignin from the cell walls, leaving pure cellulose behind. As soon as the middle lamella is attacked, the cells separate completely or cling together loosely at the corners where the thicker walls offer more resistance to the fungus. Finally the thin-walled parenchyma cells and the tracheids are entirely dissolved. The thick-walled strongly lignified wood fibers persist the longest. Only a part of them are dissolved, the remainder forming a white cellulose lining and partly filling the cavities (Fig. 15). In the final stage the cellulose lining is entirely lacking.[16]

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[15] The differences given above are much a matter of degree, however, and familiarity with the two types of decay is essential in making a reliable diagnosis.

The wood between the infected areas, as in chestnut, remains apparently as sound as ever. Here and there a hypha may be seen extending horizontally from cell to cell, but connection between the pockets is secured mainly, as in chestnut, by strands of mycelium which run radially and tangentially. As previously noted, the centers of infection have their origin between the large medullary rays, while in chestnut they may occur anywhere within the ring. Once started, however, the white areas spread and may include a portion of a compound ray within their boundaries. The decay in oak is comparable to that in chestnut except in minor respects.

The remarkable similarity which has been shown to exist superficially between the decay of oak caused by *S. frustulosum* and that of *H. rubiginosa* is even more striking when a microscopical investigation is made. The walls of the infected elements in both cases are first delignified, beginning with the tertiary layer, and pure cellulose left behind. As soon as the primary layer is reached, it is dissolved and the elements separate. Subsequently cellulose digestion goes on and the thinner-walled elements are entirely dissolved, the thicker-walled fibers disappearing last and appearing for a time as a white layer lining the cavities.

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[16] What has been said above concerning the restricted peripheral decay in chestnut applies equally well to oak.

It is of interest in this connection to compare *Stereum hirsutum* Fr. with *Hymenochaete rubiginosa* in its method of attack on wood and the chemical changes involved (Ward, 1898). This form is not so exacting as to its host as *H. rubiginosa,* but grows readily on oak, willow, horse-chestnut, pine, and other hosts. The fungus, while usually saprophytic, may spread from dead wood into the trunk and in such cases shows marked preference for the sap wood. This does not apply with *H. rubiginosa;* it is found uniformly on fallen, decorticated wood and no preference is shown for sap wood. The mycelium of *Stereum hirsutum* attacks all the elements in the diseased tissue with equal ease and localized areas of distintegration are not formed. The whole mass of tissue eventually succumbs and a general decay of the wood results. In both fungi the first evidence of decay is one of delignification. The tertiary layers are first reduced to cellulose, followed successively by the secondary and primary layers, and the swollen cellulose matrix is then consumed, layer by layer. In the case of *S. hirsutum,* the primary lamella is not attacked until the last, and before it succumbs the tertiary and secondary layers have usually entirely disappeared. This is in marked contrast to the decay caused by *H. rubiginosa,* where the middle lamella disappears as soon as lignin reduction is complete and before cellulose digestion has taken place. While closely related botanically, marked differences occur in the decay caused by the two forms.

The superficial type of decay at the periphery of the wood noted above has likewise been observed in connection with *Stereum frustulosum.*[17] The possibility still remains, however, that this may have been caused by the mycelium of another fungus as in the case of the form described in this paper. Pure cultures are necessary to decide this point.

Between the cavities the wood appears sound in both forms of decay. The pockets may enlarge occasionally and coalesce, and larger pockets thus arise. The mycelial strands accompanying the decay described here were likewise described by Hartig in connection with *Stereum frustulosum.* Both forms apparently employ the same method of attack and bring about a similar progress of decay in the wood.

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[17] Hartig, Th., loc. cit., page 19.

SUMMARY

1. *Hymenochaete rubiginosa* (Schrad.) Lév. is a common saprophyte on decorticated chestnut about Ithaca; it is found more rarely on oak.

2. The fruit bodies are annual and quite xerophytic; spores are shed intermittently during moist periods for several months.

3. Spore germination occurs best in decoctions of oak or chestnut sawdust and tap water. Mycelial growth was restricted and clamp connections were not observed.

4. The first evidence of decay in oak and chestnut consists in the formation of white spots here and there in the wood. Cavities lined with cellulose are formed through the partial or complete digestion of the elements.

5. The tissues between the infected areas remain nearly or quite as sound as in normal wood. Rarely do the pockets coalesce through the digestion of intervening tissue.

6. The chemical action of the fungus consists first in the delignification of the elements attacked. This begins with the tertiary layer and continues outward.

7. Soon after the middle lamella is attacked it is dissolved and the elements separate or remain loosely attached at the corners.

8. Cellulose digestion continues after the elements become isolated. The thin-walled elements including pith ray cells and wood parenchyma are first dissolved.

9. The pockets arising in the wood are at first lined with partially digested elements which consist of pure cellulose. In the final stage the white lining entirely disappears.

10. The decay accompanying the fungus is comparable to that caused by *Trametes abietis* Karst., on red spruce and other conifers. It has a remarkable resemblance in superficial appearance and method of attack to that caused by *Stereum frustulosum* (Pers.) Fr.

11. A superficial, peripheral type of decay, in which all the elements are attacked but not entirely digested, usually accompanies the typical decay caused by *Hymenochaete rubiginosa*. The walls remaining are dark in color and fail to respond to the cellulose reaction.

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EXPLANATION OF PLATES CXLIX–CLI

Plate CXLIX

Fig. 1. Dimidiate fruit bodies of *Hymenochaete rubiginosa* (Schrad.) Lév. on a chestnut log. × 1/9.

Fig. 2. Ellipsoidal spores of *Hymenochaete rubiginosa.*

Fig. 3. Resupinate fruit bodies on a chestnut log. × ⅔.

Fig. 4. Section of fruit body, hymenial surface above.

Fig. 5. Cross section of small chestnut log showing white areas characteristic of one stage of decay. × 8/9.

Fig. 6. Portion of the same enlarged; cavities in process of formation.

Fig. 7. Appearance of tissue in the peripheral type of decay; the hyphae are closely appressed to the cell walls and all the cells are undergoing disintegration.

Fig. 8. Germinating spores in hanging drop culture; note vacuolation in the germ tubes.

Plate CL

Fig. 9. Cross section of small chestnut log showing final stage of decay. × ⅔. The contents of the pockets have entirely disappeared.

Fig. 10. Tangential view of pockets. × ⅔. The white cellulose contents have not been dissolved.

Fig. 11. Tissue at the edge of a diseased area. Note (*a*) the delignification of the elements, (*b*) the separation due to the disappearance of the middle lamella, and (*c*) final digestion.

Fig. 12. Cross section of chestnut wood showing scattered, irregular-shaped areas where disintegration has occurred. The wood between the pockets is still sound.

Fig. 13. Tangential section of oak wood showing shape of the pockets in side view.

Fig. 14. Cross section of chestnut wood showing a portion of diseased tissue which includes one vessel.

Fig. 15. Same as figure 13. Note the partially digested, thick-walled prosenchymatous elements projecting into the cavity.

Fig. 16. Cross section of oak wood; diseased area on the extreme right (the hazy appearance in the center of the figure is not due to the attacks of the fungus).

Plate CLI

Figures 17–20 are photographs of pen drawings.

Fig. 17. Spores, germination, and germ tubes forty-eight hours after germination.

Fig. 18. Cross section of a fruit body; hymenium above.

Fig. 19. Portion of hymenium enlarged showing cystidia, basidia and spores, and immature basidia.

Fig. 20. Cross section of chestnut wood at margin of diseased area, showing sound cells at *a*, partly delignified cell at *b*, and partly digested cells at *c*.

Fig. 21. Young fruit bodies coalescing to form extensive hymenial surfaces.

Fig. 22. Cross section of chestnut wood at margin of a pocket. The shading indicates the extent of delignification in the cell walls.

Fig. 23. Wood parenchyma cells of chestnut in longitudinal view; hyphae extending horizontally through the pits.

Fig. 24. Mycelial strand extending through the tissue. These serve to connect the pockets which are subsequently formed.

Fig. 25. Pith ray and neighboring parenchyma cells in tangential view. Hyphae have passed through the pits, eroded the cell wall, and then disappeared, leaving irregular openings.

Fig. 26. Cross section of "pocket" in process of formation. The cells have been delignified and have separated from one another.

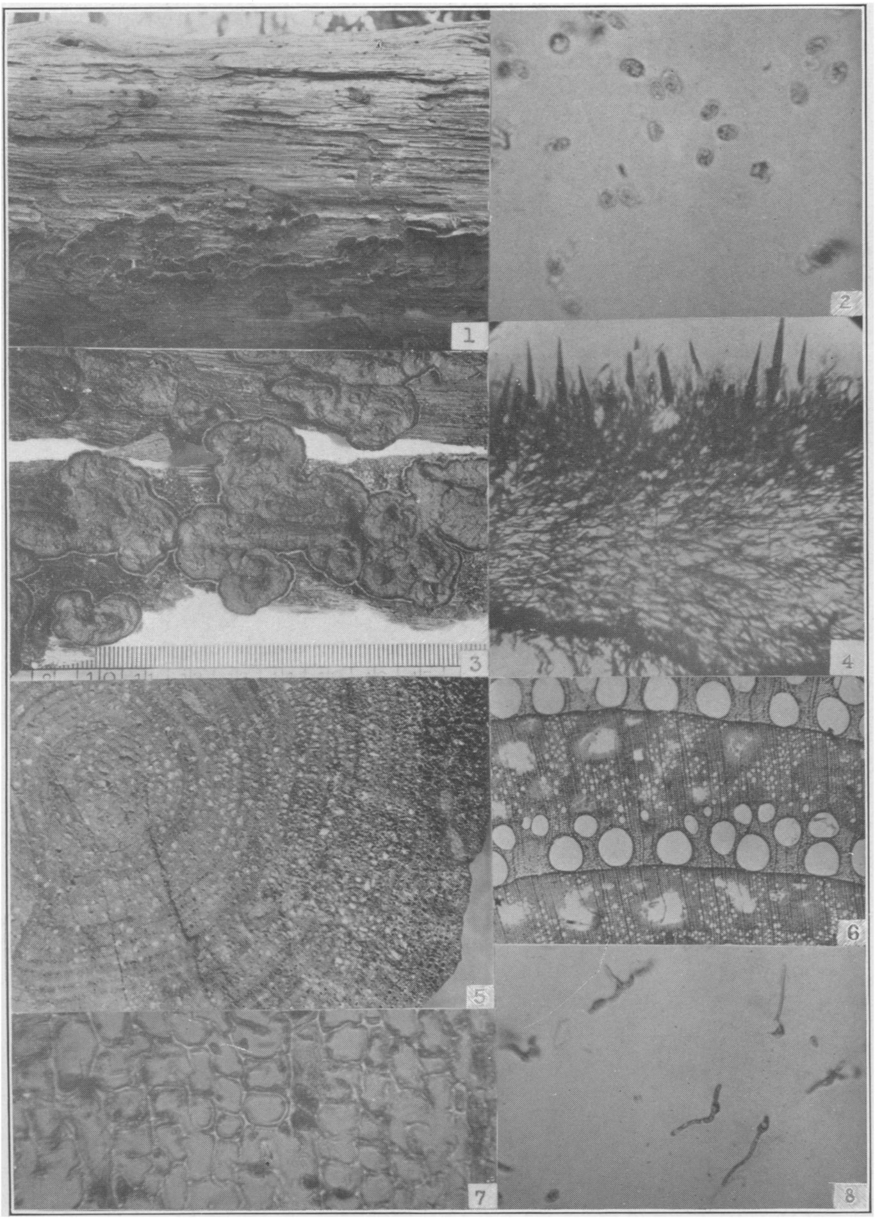
Fig. 27. Radial view of a pith ray (with other elements in the back-ground) showing the course of hyphae through the wood. Penetration of the wall occurs mainly through the pits.

Fig. 28. Cross section of chestnut log showing seasoning check. The fungus tends to follow these checks into the deeper-lying tissues.

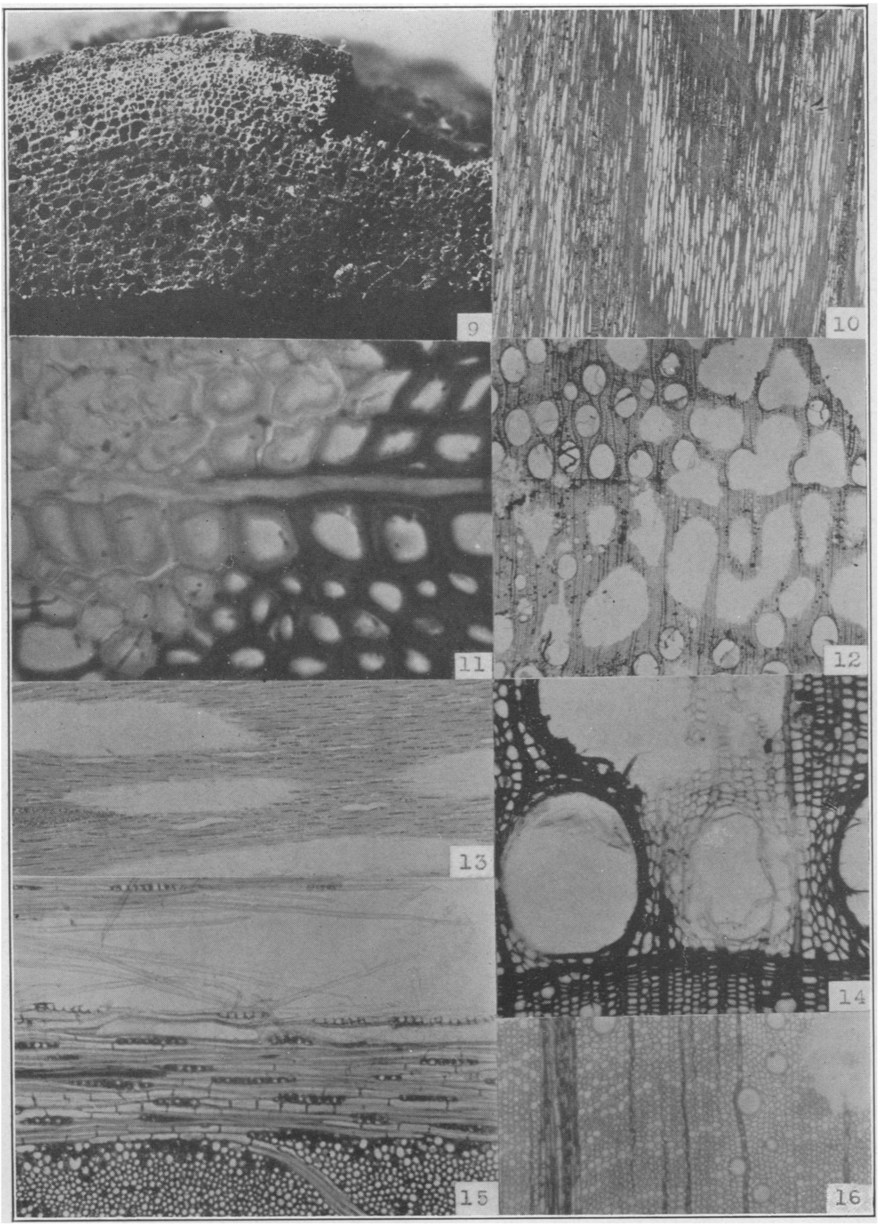
Fig. 29. Radial section of chestnut wood showing fungus filament passing through bordered pits of prosenchyma.

Fig. 30. Portion of pith ray and parenchyma cells of chestnut, tangential section. The intercellular space at (*a*) and the pits at (*b*) have been enlarged through fungal attack.

PLATE CXLIX



HYMENOCHAETE RUBIGINOSA (SCHRAD.) LÉV.



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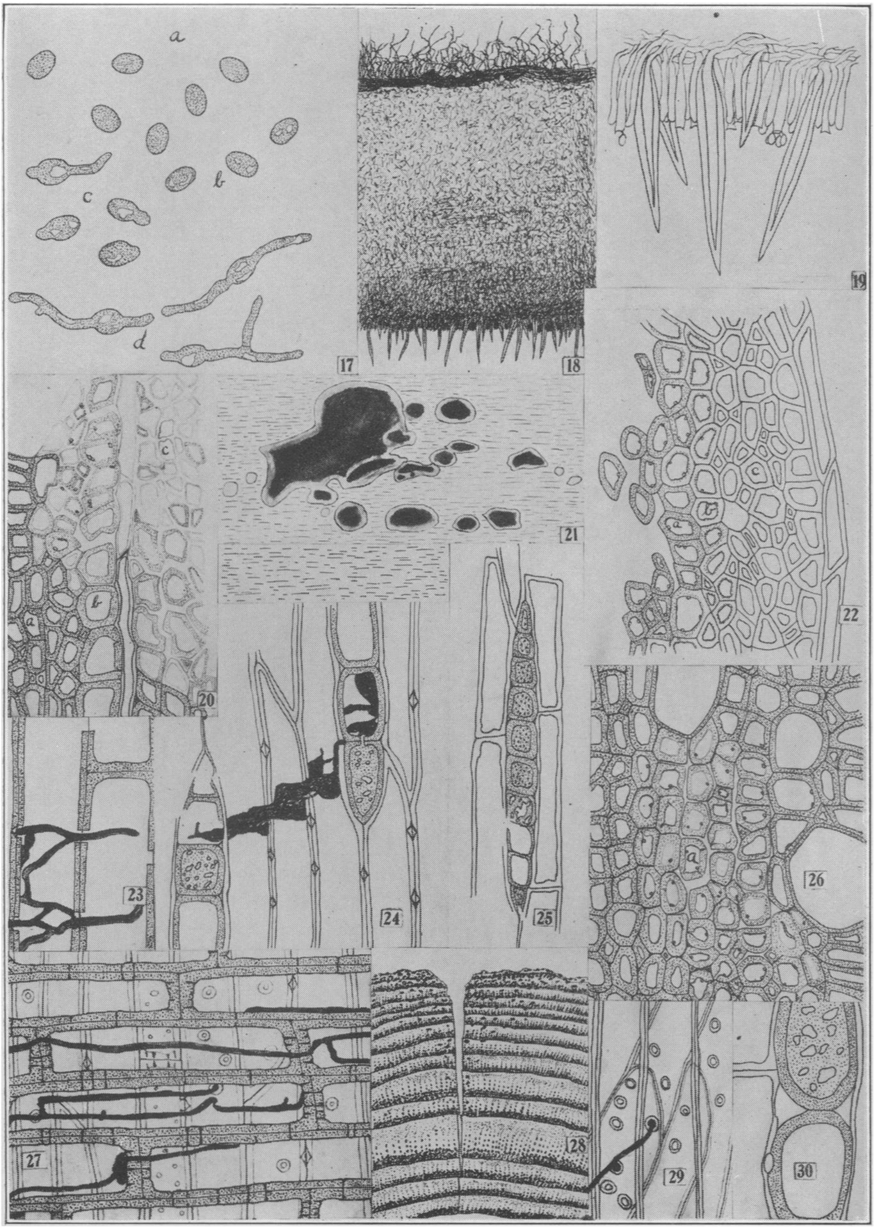

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